Changes in intrathoracic blood volume (Vval) during +Gz acceleration

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The purpose of this study was to evaluate the changes in intrathoracic blood volume during low levels of head-to-feet acceleration (+Gz). Twelve healthy male subjects trained and well-tolerated to +Gz were involved. A breathing manœuvre was applied to measure an increment of lung air volume after a 10 s voluntarily increased intrathoracic pressure (Valsalva manœuvre) upon vital capacity. This additional lung air volume (Vval) was used to represent relative changes in intrathoracic blood volume during +Gz. Blood pressure was measured by an electronic blood pressure monitor and heart rate was counted through a continuously monitored electrocardiogram. Each experimental session consisted of one control run of 8 min at +1 Gz and test run at either +2 Gz or +3 Gz at sitting position. Blood pressure and Vval were measured after 2 min of exposure and were repeated for four trials in each exposure. Since no significant changes in blood pressure and Vval were found after 2 min of exposure, mean values in steady states were presented. Vvas showed no significant variation from +1 Gz to +3 Gz, indicating that the total intrathoracic blood volume was not influenced by low levels of +Gz in sitting position. Systolic blood pressure (SBP) did not change at +2 Gz, but increased by about 10 mmHg at +3 Gz. Diastolic blood pressure (DBP) increased by about 10 mmHg and 18 mmHg at +2 Gz and +3 Gz, respectively. Heart rate (HR) increased by about 12 beats/min at +2 Gz and 40 beats/min at +3 Gz. The gradual reduction in pulse pressure (PP) and the increase in mean arterial pressure (MAP) were calculated. Increases in SBP and DBP during +Gz illustrated that there was a heart level hypertension during +Gz. The increase in heart rate was believed to be due to an activation of carotid baroreflex. This study confirms that during +Gz heart level hypertension due to active venoconstriction or increased intraabdominal pressure helps maintain intrathoracic blood volume.

Keywords: Acceleration stress, Intrathoracic blood volume.

Gravitational forces lead to a redistribution of the blood volume due to the development of considerable hydrostatic pressure in the circulatory system. Under positive G (head-to-feet acceleration, +Gz), blood drains from central circulation and pools in the dependent vascular bed. The redistribution of blood volume between the intrathoracic circulation and the capacitance vessels of the systemic circulation has been measured by X-ray technique [1]. The results indicated that the lung fields became clearer during increasing acceleration. This was assumed to be consistent with a decrease in pulmonary blood volume or a redistribution of pulmonary blood flow. A redistribution of pulmonary blood flow under +Gz had been studied extensively and a gradient increase of blood flow toward the basal component of the lung was generally agreed upon [2–4]. However, no systematic attempts have been made to measure the extent of blood in pulmonary vessels, but the evidence for its occurrence must be regarded as strong. In order to measure the changes in intrathoracic blood volume during +Gz, a breathing technique was applied to measure the increment of lung air volume which can be inspired after a brief period of voluntarily increased intrathoracic pressure (Valsalva manœuvre) upon vital capacity. This additional lung air volume, termed the Valsalva volume (Vval), could represent changes in intrathoracic blood volume because increased intrathoracic pressure expresses certain amount of blood out of the thorax and reserves some of the space for additional air [5]. Previously, the changes in Vval during orthostatic stress (footward blood pooling) induced by graded LBNP and from lying position to sitting position in our laboratory were found to be able to reflect efficiently the reduction and intrathoracic blood volume due to footward blood pooling. This volume was also found to be tightly bound to changes in cardiac output ($r = 0.603$, $P < 0.001$, unpublished data) and both variables were suggested to be modified by baroreflexes triggered by different levels of blood pooling. We applied this technique to investigate the alterations of intrathoracic blood volume during low levels of increased +Gz.

Material and methods

The study group consisted of twelve healthy male volunteers. Their characteristics are shown in Table 1. The subjects underwent thorough physical examination. The subjects were trained to operate the equipment and to perform the breathing manœuvre at various +Gz levels before the experiments began. All the subjects were able to tolerate well at least 8 min in +2 Gz. However, only four of the subjects were qualified to tolerate well 8 min in +3 Gz without untoward effects. The experiment was approved by the Internal Review Board of the University's Medical School. The subjects gave their written informed consent before participating.

The experiments were conducted in a human centrifuge (Model 81983-J-001, Rucker Control System, Oakland, CA) of the Hermann Rahn Environmental Physiology Laboratory at the State University of New York at Buffalo. The centrifuge is capable of a sustained radial acceleration, at the centre of the capsule, in the range from approximately one-tenth to ten times the acceleration due to gravity. The altitude of the capsule was controlled automatically. The floor of the capsule was always perpendicular to the vector resulting from the interaction between normal gravity and centrifugal force. The accelerations to be reported were the net resultant of these two factors. The seat was taken from an aircraft with its back inclined 15° from the vertical.

A manœuverable refilled Ohio spirometer (Ohio 822, Ohio Medical Products, Airco Inc., Medison, WI) was mounted on a stainless steel shelf fixed on to the floor of the capsule in level with the subject's mouth when seated. A 50 cm rubber tube was connected from the orifice of the spirometer to a T-shaped plastic connector. A one-way valve was inserted between the proximal end of the connector and the rubber tube to prevent air leaking back into the spirometer. An electric solenoid valve (ASCO 8210C94, Automatic Switch Co., Florham Park, NJ) was arranged on the distal end of the connector to control the opening of the airway. A mouthpiece was mounted on the middle of the connector. A voltage manometer (Model CD 15, Validyne Engineering Co., Northridge, CA) was connected with the mouthpiece in order to monitor mouth occlusion pressure. A three-channel chart recorder (Soltec 1243, Soltec Co., San Fernaudo, CA) was used to record the lung volume changes and mouth occlusion pressure. Before each experimental

Table 1. General characteristics of the subjects

Subject	Age (yr)	Weight (kg)	Height (cm)
1	23	86.4	188.0
2	21	75.0	185.0
3	34	95.5	185.4
4	24	70.5	167.6
5	20	79.5	188.0
6	21	63.6	162.6
7	23	69.5	180.3
8	20	79.5	190.5
9	20	75.0	177.8
10	21	70.9	180.3
11	22	72.7	170.2
12	22	81.8	177.8

session, the spirometer and the voltage manometer were calibrated by a 2 l syringe and a mercury manometer, respectively.

The effect of increased mouth occlusion pressure was studied by having the subjects inspire maximally. The subjects then blew against the voltage manometer, with the solenoid valve closed, rapidly raising the pressure and maintaining it for 10 s. Immediately after this procedure, an additional volume of air (Vval) was inspired maximally again and expiration was quickly performed into the spirometer. The general procedure may best be visualized from a spirographic tracing (Figure 1) record read from left to right. The maximal inspiration reached level A, at which point the subject was changed from the spirometer, by the solenoid valve, to the manometer and the pressure of the Valsalva manœuvre (horizontal tracing) was exerted. When the subject was turned back to the spirometer, additional air was inhaled rapidly and level B was reached. This was followed by complete expiration.

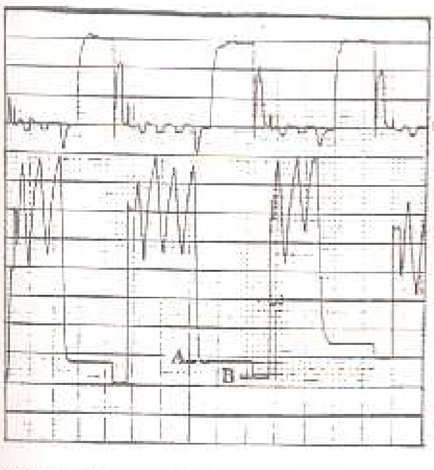

Figure 1. Spirographic tracing of the effect of a brief period of raising mouth occlusion pressure upon vital capacity. The upper tracing indicates the changes in intrapulmonary pressure and the lower tracing represents the lung volume changes. Lung volume at level A indicates that the subject inspired maximally to his vital capacity. Level B represents that an additional air volume (Vval) was inhaled after raising mouth occlusion pressure (30 mmHg) for 10 s.

In each of the following experiments, this breathing manœuvre was performed after 2 min in each situation and repeated four times, with at least 2 min rest in between. The acceptable Vval data were determined and selected if the mouth occlusion pressure was held steadily within a range of ±2 mmHg. The data were calculated to BTPS.

Blood pressure and heart rate were measured at the heart level before and during +Gz acceleration by an electronic blood pressure monitor (Infrasonade Model D4000, Puritan-Bennett Corp., Wilmington, MA). Measurements were made before each breathing manœuvre. The appearance of the subject during the proceeding of the whole experiment was monitored by a camera recorder.

Three-standard-leads EKG was monitored continually through a Grass Model 7 polygraph (Grass Medical Instruments Co., Model 7, Quincy, MA) during each exposure.

Three experimental conditions were designed from +1 Gz to +3 Gz in sitting position. During each experiment day, the control experiment was conducted at +1 Gz while sitting at rest with the centrifuge in very slow motion and the experiment at +2 Gz or +3 Gz with an onset rate of 0.9 +Gz/s was conducted after 5 min of recovery from control.

During each exposure, the subject started to perform the breathing manœuvre after 2 min of exposure. The manœuvre was repeated every 2 min, for a total of 4 trials in each +Gz exposure. Blood pressure was measured right before each breathing manœuvre. The breathing manœuvre was performed only when a stable blood pressure was confirmed and no changes in vision were reported. On any given day, measurements would be made at +1 Gz control first and then either at +2 Gz or at +3 Gz. Subjects were allowed to be exposed in one hypergravity environment only once per day and the next exposure was arranged at least two days apart. Twelve subjects were exposed to +2 Gz five times. Four subjects were exposed to +3 Gz three times.

The data for +1 Gz to +3 Gz were compared using repeated ANOVA measures. Each variable's value was evaluated as a function of time first. Since there was no significant variation in values for 2 min–8 min of exposure (steady state), the values measured during steady states in each +Gz exposure were counted for mean analysis. The Fisher-LSD analysis was applied to compare the different means among different +Gz exposures. The data for sitting at rest position served as control. The confidence level was set at 0.05.

Results

The data were collected from twelve well-trained healthy subjects exposed to 8 min of +2 Gz. Four of the twelve subjects were exposed to 8 min of +3 Gz. Each of the data presented in this article was selected when the subject was in stable condition and did not complain of any physical discomfort or changes in vision.

The mean in steady-state values of Vval, blood pressures and heart rate during +Gz are listed in Table 2. Vval showed no significant variation for +1 Gz to +3 Gz. Systolic blood pressure (SBP) did not change at +2 Gz, but increased by about 10 mmHg at +3 Gz. Diastolic blood pressure (DBP) increased by about 10 mmHg and 18 mmHg at +2 Gz and +3 Gz, respectively. Heart rate (HR) increased by about 12 beats/min at +2 Gz and 40 beats/min at +3 Gz. A gradual reduction in pulse pressure (PP) and an increase in mean arterial pressure (MAP) were calculated following the increment of +Gz.

Discussion

Positive, +Gz, acceleration produces a severe stress on the circulatory system. The magnitude of this stress imposed in the sitting position causes mainly increments of hydrostatic pressure toward the feet and, therefore, accentuates orthostatic fluid shifts from the intrathoracic compartments to the legs. It was found that increasing +Gz caused 12–50 ml/Gz of blood to pool in the legs. This initial blood pooling, which takes 25 s, is followed by a slow increase

Table 2. Blood pressure, heart rate and Vval responses during +Gz in sitting position

	+1 Gz (n = 12)	+2 Gz (n = 12)	+3 Gz (n = 4)
Vval (ml)	204.9	238.7	227.4
	10.6	32	41.9
SBP (mmHg)	121	123.9	132.0*
	2.4	2.4	4.1
DBP (mmHg)	69.9	80.5*	97.6*
	1.8	1.8	3.3
PP (mmHg)	50.9	43.9*	33.2*
	1.8	1.6	2.7
MAP (mmHg)	86.2	95.2*	109.4*
	1.9	1.7	4.0
HR (beats/min)	76.1	97.0*	115.4*
	2.3	2.3	4.1

Values represent 'mean ± SE'

SBP: Systolic blood pressure; DP: Diastolic blood pressure; PP: Pulse pressure; HR: Heart rate; MAP: Mean arterial pressure

*Significantly different from data at +1 Gz ($p < 0.05$).

**Data were calculated by the formula (PP/3) + DP. Vval. Increment of vital capacity due to Valsalva's manœuvre BTPS).

n: number of subjects participated.

in leg volume, with about 60 ml/Gz accumulating in 5 min [6, 7]. This is a modest volume compared to the 500 ml that pools in the legs in erect [8] and LBNP positions [9], and reflects probably the difference between the filling of collapsed veins and the distension of them, with venoconstriction counteracting the +Gz process [10].

Since the intrathoracic blood volume, reflected by changes in Vval, was found to be reduced by about 30–40% during high levels of LBNP (>35 mmHg) and sitting position and have a positive correlation with changes in cardiac output ($r = 0.603$, $p < 0.001$), Vval was proposed to be decreased due to the footward blood pooling during +Gz and the decrements were assumed to be paralleled to the increments of G forces. In this study, however, no significant decrease in Vval was found at +1 Gz, +2 Gz or 3 Gz. No reasonable explanation can be concluded from our study.

However, we found that SBP and DBP both increased and the increment was proportional to the increase of +Gz. The increases in MAP and decreases in PP were paralleled to the increments of +Gz (Figure 2). Lambert and Wood [11] using photokymographic recording of

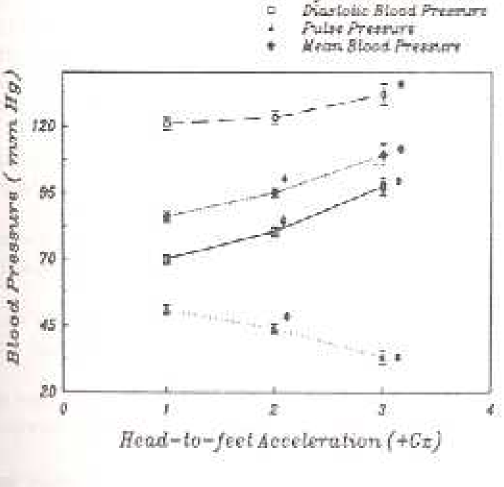

Figure 2. Blood pressure responses during +Gz. * denotes significantly different from the preceding value.

man's blood pressure in 12 healthy young men who tolerated well an acceleration of 4.5 +Gz for 15 s, showed that eye-level arterial pressure fell by about 32 mmHg for SBP and 20 mmHg for DBP. With unimpaired vision, the SBP at eye level remained above 50 mmHg. Interestingly, arterial pressure was maintained normally at the level of heart. After the initial 7 s of the exposure, a dramatic increase in arterial pressure occurred. Linnarsson and Rosenhamer [12] also found that mean arterial pressure increased by about 18 mmHg sitting at +3 Gz.

As for a reduction in venous return due to footward blood pooling, the increases in SBP and DBP at heart level and of heart rate were considered reasonably to be due to baroreflexes. However, the observed increase of heart rate cannot be attributed to the activation of baroreflex from the aortic region, since the aortic pressure was found to be increased. A decrease of arterial pressure at head level is confirmed by several studies [13–15]. The carotid sinus, no doubt in great part, was responsible for the tremendous tolerance of the circulation to long-term +Gz. Jongbloed and Noyons [16] sectioned the carotid sinus nerve and abolished the tachycardia responses to +Gz in experimental animals. Greenfield [17] also found that the compensatory rise in arterial pressure during prolonged Gz did not occur after stripping the carotid region. The physiological baroreflex triggered from carotid baroreceptors was believed to play an important role in maintaining arterial pressure at the heart level.

The reports on measurements of cardiac output changes during +Gz in human volunteers are few. Lindberg *et al.* [18] found, by dye dilution technique, that the average falls in cardiac output were 7% and 18% at +2 Gz and +3 Gz, respectively. However, a large variation in responses (+9% to –25%) between subjects was found. Arieli *et al.* [19] also reported a 19% and 22% reduction in cardiac output during prolonged +2 Gz and +3 Gz, respectively.

Since a reduction in cardiac output and an increase in mean arterial pressure were observed, a strong and enhanced total peripheral resistance was proposed. The increase in total peripheral resistance could contribute to either vasoconstriction or venoconstriction or both. Direct and indirect evidences have shown that the low-pressure system seems to behave like a container with passive elastic walls during many types of disturbances of blood volume homeostasis. In tilting a man in the upright posture or in LBNP, the distensibility of the vascular bed of the hand showed only an initial fleeting constriction or no changes [20, 21]. On the other hand, by inserting a miniature balloon technique in saphenous vein of dogs, Salzman [22] demonstrated a significant sustained increase in venomotor tone during +Gz. The same conclusion was also suggested by Newberry *et al.* [23]. They measured forearm venous compliance by a venous impeding strain gauge method in (1) low levels of +Gz, (2) LBNP of −20 mmHg and (3) 45° of head-up tilt in man. Evidence for the role of contraction of the venous reservoir in the support of the central blood volume was found only during +Gz. From these experiments, it was suggested that normal tone of the capacity vessels was high enough, at rest, to prevent excessive pooling with normal stress of +1 Gz. However, with stronger stimuli, such as high +Gz, there was a strong constriction of the capacity vessels. These findings were interpreted as evidence for the role of contraction of the venous reservoir in the support of a hypertension state of the heart under a hydrostatic load.

Another possible explanation for the increase in arterial pressure at heart level was indicated by the bottom tracing of intrarectal pressure. Intrarectal pressure was directly related to intraperitoneal pressure in the dependent regions of the abdominal cavity. This pressure was found to be increased in direct proportion to the force environment, so that at +4.5 Gz it increased five times above normal [24]. It is possible that abdominal contents behave like an anti-G suit, as if the abdominal cavity was filled with water, providing an approximate balance to the intravascular hydrostatic gradient. Therefore, venous return from the abdomen to the heart is maintained during headward acceleration, so that systematic arterial pressure was sustained as well.

In summary, the mechanism that helps maintain intrathoracic blood volume during +Gz in this study is unclear from our data. However, the increase in mean arterial pressure at heart level was found to be proportional to the increments of +Gz, which we believe are related to an active venoconstriction and a rising intraabdominal pressure. Since the pulmonary circulation was tightly bound to changes in the systemic circulation, the factors that modify system circulation are believed to play an important role also in influencing the intrathoracic blood volume. On account of these events, we believe that haemodynamic responses during +Gz were probably different in graded LBNP and while sitting, even though the footward blood pooling was the primary cause for changes in all of these.

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